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CHROMAFFIN SUBSTANCE IN RELATION TO VASOMOTOR ATAXIA, AND THE EQUILIBRIUM OF INTERNAL SECRETIONS.

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IN 1892 I called attention to a condition I denominated *vasomotor ataxia*, and suggested that this was the basis upon which various syndrome groups—Graves's disease at the one extreme and Raynaud's disease at the other extreme—were developed in accordance with the incidence of various additional extrinsic and intrinsic etiologic factors. In subsequent communications I have reported a number of cases, serving to complete the links of the chain. Among the various conditions coming under the designation suggested are those reported by Osler under the name of the "erythema group." The recent experimental observations on the production of arteriosclerosis by the injections of adrenal substance, and Wiessel's demonstration of the chromaffin system, its destruction in Addison's disease, and its defective development in arterial hypoplasia and status thymicus, are suggestive of a definite pathologic explanation of the clinical observations alluded to. The vasomotor ataxic condition may be dependent on imperfect development or inharmonious distribution of chromaffin; angina pectoris, Raynaud's disease, erythromelalgia, and intermittent claudication may be associated with excessive chromaffin development, local or general; while Graves's disease, hay fever, status thymicus, Addison's disease, acromegaly, and myxœdema are brought into relation as associated with lack of chromaffin, either from deficient development or excessive destruction. This leaves many etiologic and pathologic problems still to be solved. It brings into view once more the reciprocal relations of the thyroid, pituitary, adrenal, and thymus glands on which Sajous has laid such stress, substituting,

however, the term chromaffin system for Sajous's term of adrenal system, inasmuch as the pressure-raising substance of the adrenal is shown to be the chromaffin of which its medulla consists, and which is identical with the chromaffin distributed throughout the sympathetic system of nerves and in certain situations in the heart and vessels. In Addison's disease Wiesel has shown that chromaffin disappears both from the adrenal and the sympathetics, thus reconciling the apparently conflicting necropsy records of this disease. Leukoderma, which is often associated with vasomotor ataxia, may be found to have some relation with this group of disorders, a subject that can be better discussed when the cutaneous pigmentation of Addison's disease is brought into relation with the disappearance of chromaffin. It may be that the absorption of pigment in leukoderma is a compensatory process. At all events the metabolic balance of the internal secretions is evidently a most important matter of vital equilibrium; and the paths indicated by Sajous must be fully explored experimentally. When this is done, and exact knowledge takes the place of the speculation to which we are now, in many directions, restricted, our hygienic, therapeutic, and prophylactic resources will be much increased. At present we know empirically that thyroid gland substance is valuable in arteriosclerosis, and should be used in myxœdema, Raynaud's disease, angina pectoris, and intermittent claudication—diseases in which chromaffin may be excessive. As I have elsewhere pointed out, it belongs therapeutically with the nitrites. Perhaps a definite substance, antagonistic to chromaffin, may some day be extracted from it. We also know empirically that benefit follows the use of thymus and adrenal substance (chromaffin) in Graves's disease, hay fever, Addison's disease, and other conditions in which chromaffin is actually or relatively deficient. The subject invites investigation.